FcRn-BASED THERAPEUTICS FOR THE TREATMENT OF AUTO-IMMUNE DISORDERS

Abstract of the Disclosure

Disclosed is a transgenic knockout mouse whose genome comprises a homozygous disruption in its endogenous FcRn gene, wherein said homozygous disruption prevents the expression of a functional FcRn protein, resulting in a transgenic knockout mouse in which exogenously administered IgG1 exhibits a substantially shorter half-life, as compared to the half-life of exogenously administered IgG1 in a wild-type mouse. Also disclosed is a transgenic knockout mouse whose genome comprises a homozygous disruption in its endogenous FcRn gene, wherein said homozygous disruption prevents the expression of a functional FcRn protein, resulting in a transgenic knockout mouse which is unable to absorb maternal IgG in the prenatal or neonatal stage of development. Methods of using the transgenic knockout mouse, and cells derived therefrom, are also disclosed.